High-Dose Baclofen for Suppression of Alcohol Dependence

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To the Editor:

I WISH TO correct an inaccurate quote of my paper (Ameisen, 2005a) and to address some omissions in the paper by Garbutt and colleagues (2010) to help clarify the treatment model that I have proposed. In this model, dose-dependent *suppression* (as opposed to only *reduction)* of craving, could translate into complete and effortless suppression of dependence in alcohol-dependent patients.

The very purpose of my paper as well as of my subsequent papers (Ameisen, 2005b, 2007, 2009) is to stress that reduction of craving could be a less than therapeutic goal since it leaves patients with full-blown symptomatic alcoholism. As a consequence, patients have to fight craving so hard that relapse occurs in more than 80% of the few who succeed in becoming abstinent (Heinz et al., 2009).

Garbutt and colleagues mistakenly quote me as having reported "*profound reduction in craving*." Such a state would not have been worthy for me of reporting since reduction of craving has already been reported for 2 decades (with naltrexone, acamprosate, baclofen at 30 mg/d and topiramate). To the contrary of the above, the essence of my paper and its title describe "Complete and prolonged suppression of symptoms and consequences of AD," a phenomenon that had never been previously reported in the medical literature. I report *effortless suppression of craving* as opposed to *effort-mediated abstinence*. Using my model, others (Agabio et al., 2007; Buckam, 2007) have succeeded in replicating my results in case reports.

Garbutt and colleagues also fail to mention the rationale of my model which is that, in animals, *baclofen is the only medication shown to suppress the urge to consume alcohol*. All other anticraving medications used in the treatment of AD only *reduce* this urge (Ameisen, 2005a,b). Conversely, despite thousands of patients trialed on these drugs, suppression of craving has never been reported using naltrexone, acamprosate, topiramate, low-dose baclofen or placebo. Finally, Garbutt and colleagues' sentence "*there are case studies that suggest that larger doses of baclofen may be required for efficacy in some individuals*" misleads the reader to believe that Ameisen and Bucknam independently used high-dose (HD) baclofen in a vacuum. Yet, after having both used my model, Bucknam titled his paper: "*Suppression of* symptoms of AD and *craving* using HD baclofen" while Agabio et al. titled their paper: "Baclofen *suppresses* alcohol intake and *craving* for alcohol in a schizophrenic alcohol-dependent patient: a case report."

In a recent open-label trial (Ameisen and de Beaurepaire, 2010), baclofen has been shown to effortlessly *suppress AD* in 88% of 60 alcoholic patients. These patients no longer fit any of the DSM-IV criteria for the diagnosis of AD. The dose required ranged 60 to 300 mg/d (mean 145 mg/d).

HD baclofen has been safely used for decades at up to 300 mg/d in adults (Smith et al., 1991) and up to 180 mg/d in children (Greene, 1992) for comfort care in the treatment of muscular spasticity, a *benign condition*. At such dose, and with children being treated for up to 8 consecutive years (Greene and Fahn, 1992), there has been no report of any serious or irreversible adverse effect. By contrast, in AD, a *deadly and devastating disease*, all clinical trials conducted in the past twenty years, including that of Garbutt and colleagues, have only used the low-dose of 30 mg/d. Randomized trials should be conducted to test whether HD baclofen could altogether suppress AD as suggested in case reports as well as in an open-label trial.

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